UNITED STATES DISTRICT COURT
WESTERN DISTRICT OF WASHINGTON
AT SEATTLE

FAYE BRYANT,

Plaintiff,

v.

WYETH,

Defendant.

CASE NO. C04-1706 TSZ

ORDER

THIS MATTER comes before the Court on Defendants Wyeth LLC, f/k/a Wyeth Inc., Wyeth Pharmaceuticals, Inc. (collectively "Wyeth"), ESI Lederle, Pfizer, Inc., Pharmacia & Upjohn Company LLC ("Upjohn"), and Pharmacia Company's[1] Motion to Exclude the Testimony of Dr. Gadi, Dkt. No. 95, and Defendants' Motion to Exclude

[11] Pfizer, Inc., was dismissed as a defendant by stipulation of the parties subsequent to the filing of these motions. Docket Nos. 117 and 119.

Certain Testimony of Dr. Michaels. Dkt. No. 91. Having reviewed the memoranda, declarations, and exhibits submitted by the parties,[2] the Court enters the following order:

I. Background

This is a prescription drug product liability case in which Plaintiff, Faye Bryant, alleges that she developed breast cancer as a result of ingesting combined hormone replacement therapy (CHRT) drugs manufactured by the Defendants. CHRT consists of two medications, estrogen and progestin ("E+P"), that are prescribed in combination to treat symptoms of menopause. This case involves four drugs, Premarin, Provera, Cycrin, and Prempro. Premarin, an estrogen, Cycrin, a synthetic progestin with the generic name medroxyprogesterone acetate ("MPA"), and Prempro, an estrogen and progestin combination, are manufactured by Defendant Wyeth. Provera, an MPA, is manufactured by Defendant Pharmacia & Upjohn Company.

Mrs. Bryant alleges that she took Premarin and Provera from 1994 until 1999, and the combination drug Prempro from 2000 to 2003, to treat symptoms of menopause. Mrs. Bryant was diagnosed with breast cancer in 2004, and thereafter instituted this action on July 2, 2004. Her tumor was classified as an invasive ductal carcinoma that tested positive for estrogen and progesterone receptors (ER+/PR+). Mrs. Bryant had breast surgery, underwent radiation chemotherapy, and used the anti-estrogen drug Tamoxifen. Bryant's Third Amended Complaint asserts claims against the Defendants

[2] The Court finds that this matter can be decided on the papers submitted. Defendants' request for oral argument is therefore DENIED.

for (1) negligence under the Washington Product Liability Act (WPLA), (2) fraud, and (3) gross negligence.

Defendants move to exclude the testimony of Mrs. Bryant's specific causation experts Dr. Gadi and Dr. Michaels, alleging that neither doctor performed a reliable differential diagnosis in arriving at his opinion on causation. Both doctors submitted a supplemental declaration that addresses several of the issues that Defendants' raised in their motions to exclude. See Declaration of Dr. Gadi, Dkt. No. 132, Ex. 4; Declaration of Dr. Michaels, Dkt. No. 145, Ex. 42. Defendants' moved in their reply briefs to exclude the supplemental declarations. See Defendants' Reply Brief on Dr. Gadi at 5-6 (Dkt. No. 155) and Defendants' Reply Brief on Dr. Michaels at 6 (Dkt. No. 159).

II. Standard

Federal Rule of Evidence ("FRE") 702 controls the admissibility of expert testimony. When a party proffers an expert witness, the preliminary issue of deciding whether Rule 702 is satisfied is governed by FRE 104(a). Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 594-95 (1993). FRE 104(a) requires the proponent to establish the admissibility of the expert testimony by a preponderance of the evidence. Bourjaily v. U.S., 483 U.S. 171 (1987). In determining admissibility, the court is not bound by any of the rules of evidence, except with regard to privilege. FRE 104(a).

FRE 702 establishes two requirements for admissibility: (1) the evidence must "assist the trier of fact" either "to understand the evidence" or "to determine a fact in issue," and (2) the witness has to be sufficiently qualified to render the opinion.

> If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case. FRE 702.

The Ninth Circuit recently addressed the courts role of determining reliability under FRE 702 in Primiano v. Cook, 598 F.3d 558 (9th Cir. 2010). "For scientific opinion, the court must assess the reasoning or methodology, using as appropriate such criteria as testability, publication in peer reviewed literature, and general acceptance, but the inquiry is a flexible one." Id. at 564. The Court's inquiry as to relevance and reliability is subject to no set list of factors. See Kumho Tire v. Carmichael, 526 U.S. 137, 141-42 (1999)

The Court's focus is on the expert's methodology, not his conclusion. Metabolife Intern., Inc. v. Wornick, 264 F.3d 832, 841 (9th Cir. 2001). Once the court has assured itself that the expert's testimony "rests on a reliable foundation and is relevant to the task at hand," its gatekeeping function is satisfied. Daubert, 509 U.S. at 597. Because the trial court is "a gatekeeper, not a fact finder" U.S. v. Sandoval-Menxoza, 472 F.3d 645, 654 (9th Cir. 2006), "[s]haky but admissible evidence is to be attacked by cross examination, contrary evidence, and attention to the burden of proof, not exclusion." Primiano, 598 F.3d at 564.

III. Supplemental Declarations of Dr. Gadi and Dr. Michaels

As a preliminary matter, the Court addresses Defendants' argument that the Court should strike the supplemental declarations of Dr. Gadi and Dr. Michaels. Defendants'

argue that the Court should strike the declarations pursuant to Federal Rule of Civil Procedure (FRCP) 37(c)(1) as an attempt to improperly supplement to the experts' original disclosures of opinion. FRCP 26(a) provides that a party must disclose, as directed by the court, its expert witnesses and a report that "contain[s] a complete statement of all opinions to be expressed and the basis and reasons therefor." An expert report should be sufficiently complete as to include the substance of what the expert is expected to give in direct testimony, and the reasons for such testimony. The report should offer the "how and why" of the results, not mere conclusions. Salgado v. General Motors Corp., 150 F.3d 735, 741-42 & n. 6 (7th Cir. 1998). FRCP 37 provides, in relevant part, that a party may not rely on evidence that was not disclosed in violation of FRCP 26(a) unless the party has either a substantial justification or the information is harmless.

Defendants' cite Cueto v. Overseas Shipholding Group, Inc., 2012 WL 28357, at *2 (S.D. Cal., Jan. 4, 2012) for support. There, the defendant moved for permission to file a supplemental expert report that introduced a new legal theory and new evidence on the eve of the pretrial conference. Id. at *1. The court denied the motion, concluding that the additional information and theories should have been included in the expert's original report or in a timely supplemental report. Id. at *2.

The supplemental declarations of Dr. Gadi and Dr. Michaels do not contain "new opinions." Specifically, the declarations contain no new material information and present no opinions that were not provided to Defendants during the course of discovery. The information in the supplemental declarations relating to "ruling out" other alternative

possible causes of Mrs. Bryant's breast cancer was addressed by Dr. Gadi and Dr. Michaels during their depositions in this case. The experts' supplemental declarations merely clarify certain points in their reports in response to questions raised in Defendants' Daubert motions. Some of this clarification is in response to citations from previous testimony that are presented without context in Defendants' motions.[3]

To the extent that the supplemental declarations cite to studies not referenced in the initial expert reports, these are presented to clarify the doctors' methodology in light of the Defendants' criticism, not to present new evidence or opinions. "While Rule 26 demands that expert disclosures be "complete," there is no requirement that such disclosures cover any and every objection or criticism of which an opposing party might conceivably complain. . . . [A]n expert need not stand mute in response to an opposing party's Daubert motion." Allgood v. General Motors Corporation, 2006 WL 2669337, at *5 (S.D. Ind. 2006). Moreover, it appears that most, if not all, of the additional studies

---

[3] See, e.g., Gadi Declaration at 1-2 (Dkt. 132, Ex. 4), explaining context of statements from his May 7, 2011, deposition (Dkt. 95, Ex. 7) in a case involving a plaintiff that had a Her2New positive breast cancer.

> Mrs. Bryant's breast cancer was not Her2New positive and my testimony on the Her2New growth factors has no relevance to this case. In that prior context, defendants asked me about whether I believed it was possible to determine the cause of an individual woman's breast cancer. Understanding that defendants' use of the word "cause" refers to initiation of the first abnormality, I testified that science does not yet allow us to determine the initiating cause. Defendants took that testimony out of context to imply that I do not believe that an expert can determine the promoting cause of a cancer. That is not true. . . . As I explained at the MDL hearing, we do not usually know what caused/initiated the first precancerous cell to become abnormal. We do however know that if that cell is hormone dependent it requires hormones for growth and development.

Gadi Declaration at 1 (Dkt. 132, Ex. 4).

have previously been brought to the Defendants attention. See Report of Dr. Naftalis (Dkt. 133, Ex. 5) (citing extensively to many studies cited in Dr. Gadi's declaration). These are not supplemental reports that result in "sandbag[ging] one's opponent with claims and issues which should have been included in the expert witness' report. . . ." Lindner v. Meadow Gold Dairies, Inc., 249 F.R.D. 625, 639 (D. Haw. 2008) (quoting Beller ex rel. Beller v. United States, 221 F.R.D. 689, 695 (D. N.M. 2003) (citation omitted)). Under these circumstances, it is proper for the Court to consider the supplemental declarations when deciding the admissibility of Dr. Gadi and Dr. Michaels' expert reports. Defendants' Motion to Strike is denied.

IV. Differential Diagnosis

Defendants seek to exclude Dr. Gadi and Dr. Michaels' testimony that Mrs. Bryant's breast cancer was caused, more likely than not, by her nine years of E+P hormone therapy. Defendants do not challenge Dr. Michaels' pathological findings or his opinions about the type, hormone receptor status, stage or growth rate of Mrs. Bryant's cancer. Therefore, this order does not address that testimony.

Dr. Gadi is an oncologist and breast cancer researcher at the Fred Hutchinson Cancer Center in Seattle. He has been involved in oncologic research for over 20 years. See Gadi Curriculum Vitae (Dkt. No. 132, Ex. 1). Dr. Michaels is a pathologist focusing on breast and gynecological pathology, as well as cytopthology. He is a staff pathologist at Laboratory Medicine Consultants/Aurora Diagnostics in Las Vegas, Nevada. See Michaels Curriculum Vitae, (Dkt. No. 145, Ex. 143). Defendants' do not dispute that Dr.

Gadi and Dr. Michaels are qualified to act as experts by virtue of their knowledge, skill, experience, training, and education.[4]

To reach their conclusions about the cause of Mrs. Byrant's breast cancer, both Dr. Gadi and Dr. Michaels employed differential diagnosis.[5] To the extent that Defendants' argue that this is not a valid scientific methodology, the Court disagrees. The Ninth Circuit approved the use of differential diagnosis in Clausen v. M/V New Carissa, 339 F.3d 1049, 1057 (9th Cir. 2003) ("federal courts . . . have recognized that a properly conducted differential diagnosis is admissible under Daubert."). Moreover, the Eighth Circuit and several district courts have concluded that differential diagnosis is a valid methodology for determining the cause of breast cancer in CHRT litigation. See Scroggin v. Wyeth, 586 F.3d 547, 566 (8th Cir. 2009) cert. denied, 130 S. Ct. 3467

---

[4] Defendants' include a paragraph in the background section of their brief that could be construed as challenging Dr. Michaels' qualifications (Defendants' Motion to Exclude Michaels at 3), but do not follow their factual recitation with argument. The Court therefore concludes that Defendants' do not seriously challenge Dr. Michaels' qualifications. In any event, it appears that Dr. Michaels is qualified by virtue of his education and background in gynecological pathology and cytopthology to act as an expert in this case. See Michaels Curriculum Vitae (Dkt. No. 145, Ex. 143).

[5] The precise definition of "differential diagnosis," refers to "'the determination of which of two or more diseases with similar symptoms is the one from which the patient is suffering, by a systematic comparison and contrasting of the clinical findings.'" See Hendrix ex rel. G.P. v. Evenflo Co., Inc., 609 F.3d 1183, 1194 n.5 (11th Cir. 2010) (citing STEDMAN'S MEDICAL DICTIONARY, 428 (25th ed.1990)). However, the parties and other courts have also used the term differential diagnosis to refer to differential etiology, the "'science and study of the causes of disease and their mode of operation.'" Id.; see also Clausen v. M/V New Carissa, 339 F.3d 1049, 1057 n.4 (9th Cir. 2003) ("Whereas most physicians use the term [differential diagnosis] to describe the process of determining which of several diseases is causing a patient's symptoms . . . courts have used the term in a more general sense to describe the process by which causes of the patient's condition are identified.") Here, the Court defers to the parties and uses the term "differential diagnosis" to refer to the methodology used to determine which of two or more possible causes is responsible for Mrs. Bryant's cancer.

(2010) (upholding use of differential diagnosis as a reliable methodology for determining the cause of the plaintiff's breast cancer); see also Hines v. Wyeth, No. 04-cv-1690, 2011 WL 2680718, *4 (S.D.W. Va. Jul 08, 2011); Costa v. Wyeth, Inc., No. 04-cv-2599, 2012 WL 1069189, *3 (M.D. Fla. Mar 29, 2012); Baldonado v. Wyeth, No. 04-cv-4312, 2012 WL 1965408, *9 (N.D. Ill. May 31, 2012); Kammerer v. Wyeth, No. 04-cv-196, 2011 WL 5237754, *6 (D. Neb. Nov 01, 2011); Michael v. Wyeth, LLC, No. 04-cv-435, 2011 WL 2011480, *8 (S.D.W. Va. May 23, 2011); Rivera Adams v. Wyeth, No. 03-cv-1713, 2010 WL 5072061, at *4 (D.P.R. Dec. 3, 2010).

Although differential diagnosis may be unreliable in some circumstances, "[t]he question of whether it is reliable under Daubert is made on a case-by-case basis, focused on which potential causes should be 'ruled in' and which should be 'ruled out .'" Myers v. Illinois Central R. Co., 629 F.3d 639, 644 (7th Cir. 2010). The Court accordingly proceeds to Defendants' case-specific arguments.

The first step in completing a differential diagnosis is to compile a comprehensive list of hypotheses or causes that are generally capable of causing the patient's disease. Clausen, 339 F.3d at 1057. "After the expert rules in all of the potential hypotheses that might explain a patient's [disease], he or she must then engage in a process of elimination, eliminating hypotheses on the basis of a continuing examination of the evidence so as to reach a conclusion as to the most likely cause . . . in that particular case." Id. Here, Defendants' do not take issue with the experts' conclusion that CHRT

is a potential cause of breast cancer in post-menopausal women.[6] Rather, Defendants' argue that Drs. Gadi and Michaels' differential diagnosis should be excluded because they failed to reliably rule out several possible causes of Mrs. Bryant's breast cancer.

V. Failure to Rule Out Endogenous Hormones

Defendants' first argue that Dr. Gadi and Dr. Michaels did not reliably rule out Mrs. Bryant's endogenous hormones as a possible cause of her breast cancer. Both Dr. Gadi and Dr. Michaels testify in their expert reports that Mrs. Bryant's tumors were estrogen receptor and progesterone receptor positive (E+/P+). Gadi Expert Report at 3; Michaels Expert Report at 1. They also testify, in lay terminology, that Mrs. Bryant's tumor required the presence of estrogen and progesterone to grow from atypical pre-malignant cells into an invasive disease. See Gadi Expert Report at 4-5; Michaels Report at 2 ("In combination, based on the morphologic features of the carcinoma, the immunohistochemical results, and my review of the medical records in this case, it is clear that the female hormones estrogen and progestin played a necessary role in the growth and development of Mrs. Bryant's breast cancer."). Finally, both doctors testify that there is evidence that Bryant's own body was unable to produce sufficient endogenous hormones to cause the tumors. Gadi Expert Report at 5-6; Michaels Expert Report at 2. Defendants' argue that this analysis is unreliable, especially considering Dr.

[6] This argument has been rejected by several other courts in HT litigation. See, e.g., Hines v. Wyeth, No. 04-cv-690, 2011 WL 2680718 at *4 (S.D. W.Va. July 8, 2011) (concluding that E+P HT can be "ruled in" as a potential cause of breast cancer).

Gadi's testimony that "[r]oughtly 80 percent" of diagnosed breast cancers are hormone receptor positive.[7]

In order to "rule out" Mrs. Bryant's endogenous hormones as a cause of her breast cancer, Dr. Gadi and Dr. Michaels rely primarily on Mrs. Bryant's menopausal symptoms. Drs. Gadi and Michaels note that Mrs. Bryant reported menopausal vasomotor symptoms, including severe hot flashes and night sweats, mood swings, and vaginal dryness that are characteristic of estrogen deficiency prior to commencing CHRT. Mrs. Bryant also reported that CHRT alleviated her menopausal symptoms and that the symptoms resumed upon discontinuation of hormone therapy. Dr. Gadi and Dr. Michaels testify that Mrs. Bryant's response to the CHRT supports the conclusion that her menopausal symptoms were the result of low levels of naturally occurring hormones. The Doctors further support their analysis with evidence that following discontinuation of CHRT Mrs. Bryant's breast density decreased precipitously and her Pap smears showed vaginal atrophy.

Defendants argue that the doctors' conclusion that Mrs. Bryant did not have sufficient endogenous estrogen to "cause"[8] her breast cancer is unreliable because (1)

---

[7] Gadi Deposition at 67 (Dkt. No. 95, Ex. 6).

[8] The term "cause" produces some confusion in HT litigation like this case because the parties can use it in several different ways. Dr. Michaels detailed the different meanings in his Expert Report

> The term "cause" can have different meaning depending on the medical and scientific context. I understand the term "cause" also has a certain legal definition in the context of court proceedings. In medicine the term "cause" may be used to refer to the DNA mutational change that occurs at the cellular level as a normal breast cell becomes an abnormal cell (process of "initiation"). Additionally, the term "cause" may also be used to describe the stimulation or "promotion" of

there is no evidence of what quantity of estrogen is necessary for an ER+ breast cancer to develop, (2) there is no qualitative evidence of how much endogenous estrogen Mrs. Bryant was producing at any particular point in time, and (3) there are no published studies suggesting that women with menopausal symptoms do not have enough naturally occurring estrogen to develop ER+ breast cancer. Essentially, because endogenous (natural) and exogenous (external) estrogens are both capable of causing ER+ breast cancer, Defendants contend that the doctors' opinions ruling out Mrs. Bryant's endogenous hormones as a cause of her cancer are unreliable.

In approaching Defendants' challenge, the Court considers the operative question is not whether the doctors' considered Plaintiff's endogenous estrogen—they clearly did[9]—but rather, whether their analysis is reliable. Dr. Gadi and Dr. Michaels both explained the reasons behind their conclusion that Mrs. Bryant did not produce sufficient endogenous hormones to cause the breast cancer. Dr. Gadi explained in his deposition,

> A. Based on her symptoms, yes. [Ms. Bryant was estrogen deficient at the time that she was deciding to take hormone replacement therapy.] And certainly after she stopped, she experiences all those symptoms again."

> microscopically abnormal precancerous or cancerous cells into a clinically and histologically apparent breast cancer.

Michael Expert Report at 3. The court points this out not to suggest that one use is correct while the other is incorrect, but rather to highlight the fact that there is not a fixed meaning and we are, at times, left to guess which meaning the party intends. Here, in the context of the doctors' differential diagnosis, the term "cause" is used to refer to the promotion or aggravation of precancerous atypical cells into a diagnosable breast cancer.

[9] Gadi Expert Report at 6 ("Mrs. Bryant did not produce endogenous hormones sufficiently to avoid menopause symptoms[.]"); Michaels Expert Report at 3 ("There were only two possible sources of the female hormones necessary to develop and grow her hormone-dependent breast cancer: endogenous hormones and exogenous hormones.").

> Q. And is that understanding as you've just expressed it, based on your education, experience as an oncologist, in addition to your understanding as gleaned from the medical literature that's available?
>
> A. Correct. And, you know, we talked about her symptoms. But also clinically the disappearance of density in her mammograms, the atrophy in her vaginal tissues, all of those are -- the totality there suggests that she was estrogen deficient.

Gadi Bryant Deposition at 131-32 (Response, Ex. 8). This is similar to Dr. Michaels' deposition testimony that Mrs. Bryant's symptoms of menopause, as well as her clinical markers for breast density and vaginal tissue, indicated that she was estrogen deficient.

> Q. Are you basing your opinion regarding her endogenous hormone levels on -- solely on the presence of the atrophic pattern seen on her Pap smear at or near the time of her breast cancer diagnosis?
>
> . . . .
>
> A. Well, that's one aspect, but her symptoms that she had complained about prior to starting HRT of hot flashes, night sweats, mood swings, and vaginal dryness reportedly worsened after she was withdrawn from HRT by Dr. McGill.

Michaels Bryant Deposition at 58 (Response, Ex. 52).

Addressing a similar Daubert challenge, the court in Scroggin rejected the Defendants' contention that Dr. Naftalis' testimony that plaintiff's endogenous hormones could be "ruled out" as a cause of her cancer was unreliable. Scroggin, 586 F.3d 566-67 & n.12. There, the court found that Naftalis' testimony was admissible.

> Knowing that Scroggin's breast cancer was hormone dependent, Dr. Naftalis's differential diagnosis sought to determine the cause of Scroggin's breast cancer by ruling out the two possible sources of these hormones: (1) Scroggin produced the hormones herself, or (2) they came from the hormone replacement therapy she had taken for the past eleven years.

> Scroggin presented evidence that her menopausal symptoms were relieved by hormone replacement therapy, confirming that her own body was unable to produce sufficient hormones and therefore could not be the cause. The remaining source was the combination of Premarin, Provera, and Prempro. Accordingly, Scroggin presented evidence establishing a causal link between breast cancer and estrogen plus progestin use, particularly for the length of time Scroggin was taking the drugs.

Scroggin, 586 F.3d at 566. This analysis has been adopted by other courts in HT litigation. See Hines v. Wyeth, No. 04-cv-690, 2011 WL 2680718 at *6 (S.D. W.Va. July 8, 2011); Baldonado v. Wyeth, No. 04-cv-4312, 2012 WL 1964480 (N.D. Illinois May 31, 2012). The court adopts this analysis here.

Here, Defendants' also argue that the experts' testimony should be excluded because neither doctor identified what amount of hormones are necessary to cause ER+ breast cancer or what amount of endogenous hormones were naturally present in Mrs. Bryant. These critiques of the experts' testimony properly go to weight. A district court is justified in excluding evidence if an expert "utterly fails . . . to offer an explanation for why the proffered alternative cause" was ruled out. Cooper v. Smith & Nephew, Inc., 259 F.3d 194, 202 (4th Cir. 2001). The expert must provide reasons for rejecting alternative hypotheses "using scientific methods and procedures" and the elimination of those hypotheses must be founded on more than "subjective beliefs or unsupported speculation." Claar v. Burlington N. R.R. Co., 29 F.3d 499, 502 (9th Cir. 1994). Here, Defendants' point to perceived weaknesses in the experts' analysis. But they do not effectively argue that Dr. Gadi and Dr. Michaels failed to offer an explanation for why the alternative cause was ruled out or demonstrate that the doctors' ruled out endogenous

hormones based merely on subjective belief or unsupported speculation. The Court is confident that any weakness in the experts' testimony can be explored at trial.

Further, the failure to identify published studies stating that women with menopausal symptoms do not have enough endogenous estrogen to develop ER+ cancer is also not a sufficient reason to exclude the doctors' testimony under these circumstances. The factors outlined in Daubert "do not constitute a definitive checklist or test." Kumho, 526 U.S. at 150 (internal quotations omitted). "[T]he case law specific to differential diagnosis recognizes that the absence of peer-reviewed studies does not in itself prevent an expert" from reaching a reliable conclusion as to causation. Clausen, 339 F.3d at 1060-61 (concluding that lack of published studies supporting diagnostic hypotheses did not make expert's opinion "junk science"). Rather, the district court has "broad latitude when it decides how to determine reliability." Id.

VI. Failure to Rule Out Obesity and Family History

Defendants next argue that Dr. Gadi and Dr. Michaels did not reliably rule out body weight and family history as risk factors for breast cancer in Mrs. Bryant's case. With respect to Dr. Gadi, he discussed in his report both that Mrs. Bryant was overweight and that her sister also developed post-menopausal breast cancer, but ruled them out as causes of her breast cancer. Dr. Michaels did not specifically address risk factors other than endogenous and exogenous hormones in his report, but did address them in his deposition testimony.

As an initial matter, Defendants' argument is unconvincing. Mrs. Bryant's theory of causation is that she developed ER+/PR+ breast cancer that required the presence

hormones in order to grow. Her theory is one of promotion, not initiation. See Baldonado, 2012 WL 1965408 at *17. Thus, both Dr. Gadi and Dr. Michaels testified primarily about the possible sources of sufficient hormones to promote Mrs. Bryant's hormone-dependent tumor. See Scroggin, 586 F.3d at 567 (observing that Dr. Naftalis' discussion of "possible risk factors" was "not necessary to the formation" of her opinion on the source of hormones necessary for the promotion of the plaintiff's breast cancer). But even assuming that a reliable differential diagnosis requires the expert to address every possible alternative cause under these circumstances, both Dr. Gadi and Dr. Michaels addressed and dismissed obesity and family history as a possible sole cause of Mrs. Bryant's cancer.

With regard to obesity, Dr. Gadi explained in his report that

> endogenous hormone production from fat tissues can be eliminated as a cause of breast cancer in Mrs. Bryant. . . . Had Mrs. Bryant been a non-user of CHRT, her mass would have been associated with increased breast cancer risk compared to thin women. However, it is important to note that in CHRT users, the promoter effect of her BMI would not have been statistically linked with breast cancer (Berstad et al., 2010; Moimoto et al., 2002). Expressed differently, regardless of her BMI, CHRT use would have been overwhelmingly more causal at promoting her breast cancer.

Gadi Expert Report at 6. Dr. Gadi also noted in his report that Mrs. Bryant's sister was diagnosed with breast cancer. However, he concluded that this family history was not a compelling risk factor because "her sister was post-menopausal and on hormone replacement therapy at the time of her diagnosis arguing strongly against and [sic] inherited genetic component such as mutant BRCA1 or BRCA2 genes." Gadi Expert Report at 2.

With respect to Dr. Michaels, Defendants are correct that he does not directly address any of Mrs. Bryant's risk factors beyond exogenous and endogenous hormones in his report. In considering her case, he "focused on identifying the source of the hormones necessary to develop and grow her hormone-dependent breast cancer." Michaels Declaration at 1 (Response, Ex. 42). He concluded that "none of Mrs. Bryant's other risk factors could have provided a sufficient source of hormones needed for growth and development of her hormone receptor positive breast cancer." Id. As discussed above, this analysis is sufficient to pass the threshold Daubert analysis for differential diagnosis.

However, as Dr. Michaels explained in his supplemental declaration, he did review her entire case and analyze each of her risk factors including obesity and family history. Michaels Declaration at 1 (Response, Ex. 42); see also Michaels Bryant Deposition at 42-47, 73-75 (Response, Ex. 52). For example, Dr. Michaels was questioned at length about the impact of Mrs. Bryant's weight on her production of endogenous hormones.

> Q. What were Ms. Bryant's risk factors for breast cancer?
>
> . . . .
>
> A. I believe that, again, just looking at general epidemiology, not necessarily in the setting of HRT, but had she not been on HRT, her BMI, which was elevated.
>
> . . . .
>
> Q. Would her BMI be a risk factor for breast cancer after she stopped taking hormone therapy, in your opinion?

> A. I would say that, given her particular case, which is what we're talking about, that it was likely, given the size of the tumor and the overall appearance with the presence of atypical ductal hyperplasia and the presence of metastasis in a lymph node, that she likely already had invasive ductal carcinoma by the time she went of HRT. But I would say that the presence of a high BMI or a possibly obese woman would be an additional promoter in the absence of HRT.

Michaels Bryant Deposition at 42-44 (Response, Ex. 52). This dialogue indicates that Dr. Michaels considered obesity an additional risk factor and that he ruled it out given the particular circumstances of this case. Dr. Michaels also identified that Mrs. Bryant's sister's diagnosis with breast cancer was an additional risk factor, but concluded that the risk was not conclusive where, as here, the sister was also post-menopausal and on HT. Michaels Bryant Deposition at 74-75 (Response, Ex. 52) (stating that a first degree relative with breast cancer is a risk factor, but that each individual case needs to be addressed independently, such as the age of the relative at diagnosis).

An expert must provide reasons for rejecting alternative. Claar, 29 F.3d at 502. Here, both Dr. Gadi and Dr. Michaels have provided the reasons they rejected the possible alternative causes of obesity and family history in Mrs. Bryant's case.

VII. Conclusion

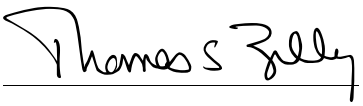
The Court is satisfied that Dr. Gadi and Dr. Michaels' testimony is admissible. Defendants' challenges go to the weight, not the admissibility, of the doctors' specific causation testimony. Defendants may, if otherwise appropriate, explore Dr. Gadi and Dr. Michaels' analysis of the alternative risk factors on cross-examination at trial. See In re Prempro Prods. Liab. Litig., Nos. 03-cv-1507, 05-cv-163, 2006 WL 2414062, at *3 (E.D. Ark. Aug. 21, 2006) ("[W]hile both reports are somewhat conclusive, rather than

explanatory, I cannot say that either expert used improper methodology. . . . Their conclusions can be tested during cross-examination.").

For the foregoing reasons, the Court finds that Dr. Gadi and Dr. Michaels' differential diagnosis testimony is reliable and satisfies the requirements of Rule 702 and Daubert v, Merrell Dow Pharm., Inc., 509 U.S. 579 (1993). Defendants' motions, Dkt. Nos. 91 and 95, are DENIED.

IT IS SO ORDERED.

Dated this 19th day of July, 2012.



THOMAS S. ZILLY
United States District Judge